WEST Search History

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DATE: Friday, August 18, 2006

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L8	L1 and 424/208.1.ICLS.	1
☐	L7	L2 and 424/208.1.ICLS.	0
☐	L6	L5 and CCR5	1
☐	L5	L3 and 530/388.22.ICLS.	18
☐	L4	L3 and 435/7.8.ICLS.	12
☐	L3	L1 and 435/7.24.ICLS.	225
☐	L2	L1and 435/7.24.ICLS.	0
☐	L1	435/7.21.ICLS.	2310

END OF SEARCH HISTORY

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=> pseudotype
L34       1336 PSEUDOTYPE

=> HIV
L35     201158 HIV

=> "macrophageg tropic"
L36          0 "MACROPHAGEG TROPIC"

=> "M tropic"
L37        466 "M TROPIC"

=> L35 and L37
L38        455 L35 AND L37

=> L34 and L38
L39          7 L34 AND L38

=> D L39 IBIB ABS 1-7

L39  ANSWER 1 OF 7  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2004:743311  CAPLUS
DOCUMENT NUMBER:          141:253793
TITLE:                    Inhibition of human immunodeficiency virus type 1
                          replication by Z-100, an immunomodulator extracted
                          from human-type tubercle bacilli, in macrophages
AUTHOR(S):                Emori, Yutaka; Ikeda, Tamako; Ohashi, Takashi; Masuda,
                          Takao; Kurimoto, Tadashi; Takei, Mineo; Kannagi, Mari
CORPORATE SOURCE:         Department of Immunotherapeutics, Graduate School,
                          Tokyo Medical and Dental University, Tokyo, 113-8519,
                          Japan
SOURCE:                   Journal of General Virology (2004), 85(9), 2603-2613
                          CODEN: JGVIAY; ISSN: 0022-1317
PUBLISHER:                Society for General Microbiology
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB    Z-100 is an arabinomannan extracted from Mycobacterium tuberculosis that has
      various immunomodulatory activities, such as the induction of interleukin
      12, interferon gamma (IFN-γ) and β-chemokines.  The effects of
      Z-100 on human immunodeficiency virus type 1 (HIV-1) replication
      in human monocyte-derived macrophages (MDMs) are investigated in this
      paper.  In MDMs, Z-100 markedly suppressed the replication of not only
      macrophage-tropic (M-tropic) HIV-1 strain (
      HIV-1JR-CSF), but also HIV-1 pseudotypes that
      possessed amphotropic Moloney murine leukemia virus or vesicular
      stomatitis virus G envelopes.  Z-100 was found to inhibit HIV-1
      expression, even when added 24 h after infection.  In addition, it
      substantially inhibited the expression of the pNL43lucΔenv vector
      (in which the env gene is defective and the nef gene is replaced with the
      firefly luciferase gene) when this vector was transfected directly into
      MDMs.  These findings suggest that Z-100 inhibits virus replication,
      mainly at HIV-1 transcription.  However, Z-100 also
      downregulated expression of the cell surface receptors CD4 and CCR5 in
      MDMs, suggesting some inhibitory effect on HIV-1 entry.  Further
      expts. revealed that Z-100 induced IFN-β production in these cells,
      resulting in induction of the 16-kDa CCAAT/enhancer binding protein
      (C/EBP) β transcription factor that represses  HIV-1 long
      terminal repeat transcription.  These effects were alleviated by SB
      203580, a specific inhibitor of p38 mitogen-activated protein kinases
      (MAPK), indicating that the p38 MAPK signaling pathway was involved in
      Z-100-induced repression of HIV-1 replication in MDMs.  These
      findings suggest that Z-100 might be a useful immunomodulator for control
      of HIV-1 infection.
REFERENCE COUNT:          58    THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS
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RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L39 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:135341 CAPLUS
DOCUMENT NUMBER: 134:294410
TITLE: Use of helper-free replication-defective simian immunodeficiency virus-based vectors to study macrophage and T tropism: evidence for distinct levels of restriction in primary macrophages and a T-cell line
AUTHOR(S): Kim, Steve S.; You, Xue Juan; Harmon, Mary-Elizabeth; Overbaugh, Julie; Fan, Hung
CORPORATE SOURCE: Department of Molecular Biology and Biochemistry, University of California at Irvine, Irvine, CA, 92697, USA
SOURCE: Journal of Virology (2001), 75(5), 2288-2300
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Cell tropism of human and simian immunodeficiency viruses (HIV and SIV, resp.) is governed in part by interactions between the viral envelope protein and the cellular receptors. However, there is evidence that envelope-host cell interactions also affect postentry steps in viral replication. We used a helper-free replication-defective SIV macaque (SIVmac)-based retroviral vector carrying the enhanced jellyfish green fluorescent protein inserted into the nef region (V1EGFP) to examine SIV tropism in a single cycle of infection. Vector stocks containing envelope proteins from three different SIVmac clones, namely, SIVmac239 (T-lymphocyte tropic [T-tropic]), SIVmac316 (macrophage tropic [M -tropic]), and SIVmac1A11 (dualtropic), were tested. SIVmac239 replicates efficiently in many human T-cell lines, but it does not efficiently infect primary rhesus macrophages. Conversely, SIVmac316 efficiently infects primary macrophages, but it does not replicate in Molt4-Clone8 (M4C8) T cells. SIVmac1A11 replicates efficiently in both cell types. When primary macrophages were infected with V1EGFP pseudotyped by SIVmac316 or SIVmac1A11 envelopes, the infection was substantially (ca. 200- to 300-fold) more efficient than for the SIVmac239 pseudotype. Thus, in primary macrophages, a major component of M vs. T tropism involves relatively early events in the infection cycle. Quant. PCR studies indicated that synthesis and transport of vector DNA into the nucleus were similar for macrophages infected with the clone 239 and 316 pseudotypes, suggesting that the restriction for SIVmac239 infection is after reverse transcription and nuclear import of viral DNA. When the same vector pseudotypes were used to infect M4C8 cells, they all showed approx. equivalent infectivities, even though replication-competent SIVmac316 does not continue to replicate in these cells. Therefore, in M4C8 cells, restriction involves a late step in the infection cycle (after proviral integration and expression). Thus, depending on the cell type infected, envelope-dependent cell interactions that govern SIV M and T tropism may involve different steps in infection.
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L39 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:217653 CAPLUS
DOCUMENT NUMBER: 133:2379
TITLE: A block to human immunodeficiency virus type 1 assembly in murine cells
AUTHOR(S): Mariani, Roberto; Rutter, Gabriel; Harris, Matthew E.; Hope, Thomas J.; Krausslich, Hans-Georg; Landau, Nathaniel R.
CORPORATE SOURCE: Infectious Disease Laboratory, The Salk Institute for Biological Studies, La Jolla, CA, 92037, USA

SOURCE: Journal of Virology (2000), 74(8), 3859-3870
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Human immunodeficiency virus type 1 (HIV-1) does not replicate in murine cells. The basis of this block was investigated by infecting a murine NIH 3T3 reporter cell line that stably expressed human CD4, CCR5, and cyclin T1 and contained a transactivatable HIV-1 long terminal repeat (LTR)-green fluorescent protein (GFP) cassette. Although the virus entered efficiently, formed provirus, and was expressed at a level close to that in a highly permissive human cell line, the murine cells did not support M-tropic HIV-1 replication. To determine why the virus failed to replicate, the efficiency of each postentry step in the virus replication cycle was analyzed using vesicular stomatitis virus G pseudotypes. The murine cells supported reverse transcription and integration at levels comparable to those in the human osteosarcoma-derived cell line GHOST.R5, and human cyclin T1 restored provirus expression, consistent with earlier findings of others. The infected murine cells contained nearly as much virion protein as did the human cells, but released less than 1/500 the amount of p24gag into the culture medium. A small amount of p24gag was released and was in the form of fully infectious virus. Electron microscopy suggested that aberrantly assembled virion protein had accumulated in cytoplasmic vesicular structures. Virions assembling at the cell membrane were observed but were rare. The entry of M-tropic JR.FL-pseudotyped reporter virus was moderately reduced in the murine cells, suggesting a minor reduction in coreceptor function. A small reduction in the abundance of full-length viral mRNA transcripts was also noted; however, the major block was at virion assembly. This could have been due to a failure of Gag to target to the cell membrane. This block must be overcome before a murine model for HIV-1 replication can be developed.

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L39 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:336092 CAPLUS
DOCUMENT NUMBER: 131:128926
TITLE: CD4 receptor-dependent entry of human immunodeficiency virus type-1 env-pseudotypes into CCR5-, CCR3-, and CXCR4-expressing human alveolar macrophages is preferentially mediated by the CCR5 coreceptor
AUTHOR(S): Park, In-Woo; Koziel, Henry; Hatch, William; Li, Xiuhong; Du, Bin; Groopman, Jerome E.
CORPORATE SOURCE: Divisions of Experimental Medicine, Hematology/Oncology, and Pulmonary and Critical Care Medicine, Department of Medicine, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02115, USA
SOURCE: American Journal of Respiratory Cell and Molecular Biology (1999), 20(5), 864-871
CODEN: AJRBEL; ISSN: 1044-1549
PUBLISHER: American Lung Association
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Alveolar macrophages (AM) are important host-defense cells and targets of human immunodeficiency virus type 1 (HIV-1) infection. However, the receptors mediating HIV-1 entry into AM are not completely characterized. The authors observed that, in addition to CD4 receptors, AM from healthy adults expressed low levels of CCR5, CCR3, and CXCR4 chemokine receptors by flow cytometry, and specific mRNA was detected for all three

receptors by reverse transcriptase/polymerase chain reaction. The macrophage monocytotropic (M-tropic; YU2) and dual-tropic (89.6) HIV-1 env-pseudotypes entered AM efficiently, as expected given CCR3 and CCR5 expression. However, the T-lymphocytotropic (T-tropic; HXB2) pseudotype did not enter AM despite expression of the appropriate chemokine coreceptor CXCR4. Incubation of AM with regulated on activation, normal T cells expressed and secreted (RANTES) significantly impaired entry of the M-tropic (YU2) HIV-1 pseudotype, whereas SDF-1β or eotaxin did not impair entry. The entry of simian immunodeficiency virus (SIV) pbj1.9 env-pseudotype into AM was not blocked by RANTES, SDF-1β, or eotaxin. The competence of these chemokine receptors for virus entry was confirmed in Cf2Th canine thymocytes cotransfected with the human CD4 and chemokine receptors. Entry of the M-tropic (YU2) HIV-1 pseudotype was shown to be mediated by either CCR3 or CCR5, the T-tropic (HXB2) HIV-1 pseudotype by CXCR4, and the dual-tropic (89.6) HIV-1 or the SIVpbj1.9 pseudotype by CCR5, CCR3, or CXCR4. The data indicate that the mechanisms for HIV-1 entry are both receptor-specific and cell type-specific, and that chemokine receptor expression on AM does not fully explain cell susceptibility to different virus isolates.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L39 ANSWER 5 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2001:187680 BIOSIS

DOCUMENT NUMBER: PREV200100187680

TITLE: Use of helper-free replication-defective simian immunodeficiency virus-based vectors to study macrophage and T tropism: Evidence for distinct levels of restriction in primary macrophages and a T-cell line.

AUTHOR(S): Kim, Steve S.; You, Xue Juan; Harmon, Mary-Elizabeth; Overbaugh, Julie; Fan, Hung [Reprint author]

CORPORATE SOURCE: Department of Molecular Biology and Biochemistry, University of California, Irvine, 3221 Biological Sciences II, Irvine, CA, 92697-3905, USA
hyfan@uci.edu

SOURCE: Journal of Virology, (March, 2001) Vol. 75, No. 5, pp. 2288-2300. print.
CODEN: JOVIAM. ISSN: 0022-538X.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 20 Apr 2001
Last Updated on STN: 18 Feb 2002

AB Cell tropism of human and simian immunodeficiency viruses (HIV and SIV, respectively) is governed in part by interactions between the viral envelope protein and the cellular receptors. However, there is evidence that envelope-host cell interactions also affect postentry steps in viral replication. We used a helper-free replication-defective SIV macaque (SIVmac)-based retroviral vector carrying the enhanced jellyfish green fluorescent protein inserted into the nef region (V1EGFP) to examine SIV tropism in a single cycle of infection. Vector stocks containing envelope proteins from three different SIVmac clones, namely, SIVmac239 (T-lymphocyte tropic (T-tropic)), SIVmac316 (macrophage tropic (M -tropic)), and SIVmac1A11 (dualtropic), were tested. SIVmac239 replicates efficiently in many human T-cell lines, but it does not efficiently infect primary rhesus macrophages. Conversely, SIVmac316 efficiently infects primary macrophages, but it does not replicate in Molt4-Clone8 (M4C8) T cells. SIVmac1A11 replicates efficiently in both cell types. When primary macrophages were infected with V1EGFP pseudotyped by SIVmac316 or SIVmac1A11 envelopes, the infection was substantially (ca. 200- to 300-fold) more efficient than for the SIVmac239 pseudotype. Thus, in primary macrophages, a major component of M

versus T tropism involves relatively early events in the infection cycle. Quantitative PCR studies indicated that synthesis and transport of vector DNA into the nucleus were similar for macrophages infected with the clone 239 and 316 pseudotypes, suggesting that the restriction for SIVmac239 infection is after reverse transcription and nuclear import of viral DNA. When the same vector pseudotypes were used to infect M4C8 cells, they all showed approximately equivalent infectivities, even though replication-competent SIVmac316 does not continue to replicate in these cells. Therefore, in M4C8 cells, restriction involves a late step in the infection cycle (after proviral integration and expression). Thus, depending on the cell type infected, envelope-dependent cell interactions that govern SIV M and T tropism may involve different steps in infection.

L39 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:229706 BIOSIS
DOCUMENT NUMBER: PREV200000229706
TITLE: A block to human immunodeficiency virus type 1 assembly in murine cells.
AUTHOR(S): Mariani, Roberto; Rutter, Gabriel; Harris, Matthew E.; Hope, Thomas J.; Kraeusslich, Hans-Georg; Landau, Nathaniel R. [Reprint author]
CORPORATE SOURCE: Infectious Disease Laboratory, The Salk Institute for Biological Studies, 10010 N. Torrey Pines Rd., La Jolla, CA, 92037, USA
SOURCE: Journal of Virology, (April, 2000) Vol. 74, No. 8, pp. 3859-3870. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Jun 2000
Last Updated on STN: 5 Jan 2002

AB Human immunodeficiency virus type 1 (HIV-1) does not replicate in murine cells. We investigated the basis of this block by infecting a murine NIH 3T3 reporter cell line that stably expressed human CD4, CCR5, and cyclin T1 and contained a transactivatable HIV-1 long terminal repeat (LTR)-green fluorescent protein (GFP) cassette. Although the virus entered efficiently, formed provirus, and was expressed at a level close to that in a highly permissive human cell line, the murine cells did not support M-tropic HIV-1 replication. To determine why the virus failed to replicate, the efficiency of each postentry step in the virus replication cycle was analyzed using vesicular stomatitis virus G pseudotypes. The murine cells supported reverse transcription and integration at levels comparable to those in the human osteosarcoma-derived cell line GHOST.R5, and human cyclin T1 restored provirus expression, consistent with earlier findings of others. The infected murine cells contained nearly as much virion protein as did the human cells but released less than 1/500 the amount of p24gag into the culture medium. A small amount of p24gag was released and was in the form of fully infectious virus. Electron microscopy suggested that aberrantly assembled virion protein had accumulated in cytoplasmic vesicular structures. Virions assembling at the cell membrane were observed but were rare. The entry of M-tropic JR.FL-pseudotyped reporter virus was moderately reduced in the murine cells, suggesting a minor reduction in coreceptor function. A small reduction in the abundance of full-length viral mRNA transcripts was also noted; however, the major block was at virion assembly. This could have been due to a failure of Gag to target to the cell membrane. This block must be overcome before a murine model for HIV-1 replication can be developed.

L39 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1999:309703 BIOSIS
DOCUMENT NUMBER: PREV199900309703
TITLE: CD4 receptor-dependent entry of human immunodeficiency

virus type-1 env-pseudotypes into CCR5-, CCR3-, and CXCR4-expressing human alveolar macrophages is preferentially mediated by the CCR5 coreceptor.

AUTHOR(S): Park, In-Woo; Koziel, Henry; Hatch, William; Li, Xiuhong; Du, Bin; Groopman, Jerome E. [Reprint author]

CORPORATE SOURCE: Div. of Experimental Medicine, Harvard Institutes of Medicine, Beth Israel Deaconess Medical Center, 4 Blackfan Circle, 3rd Floor, Boston, MA, 02115, USA

SOURCE: American Journal of Respiratory Cell and Molecular Biology, (May, 1999) Vol. 20, No. 5, pp. 864-871. print.
CODEN: AJRBEL. ISSN: 1044-1549.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 17 Aug 1999
Last Updated on STN: 17 Aug 1999

AB Alveolar macrophages (AM) are important host-defense cells and targets of human immunodeficiency virus type 1 (HIV-1) infection. However, the receptors mediating HIV-1 entry into AM are not completely characterized. We observed that, in addition to CD4 receptors, AM from healthy adults expressed low levels of CCR5, CCR3, and CXCR4 chemokine receptors by flow cytometry, and specific messenger RNA was detected for all three receptors by reverse transcriptase/polymerase chain reaction. The macrophage monocytotropic (M-tropic; YU2) and dual-tropic (89.6) HIV-1 env-pseudotypes entered AM efficiently, as expected given CCR3 and CCR5 expression. However, the T-lymphocytotropic (T-tropic; HXB2) pseudotype did not enter AM despite expression of the appropriate chemokine coreceptor CXCR4. Incubation of AM with regulated on activation, normal T cells expressed and secreted (RANTES) significantly impaired entry of the M-tropic (YU2) HIV-1 pseudotype, whereas SDF-1beta or eotaxin did not impair entry. The entry of simian immunodeficiency virus (SIV) pbj1.9 env-pseudotype into AM was not blocked by RANTES, SDF-1beta, or eotaxin. The competence of these chemokine receptors for virus entry was confirmed in Cf2Th canine thymocytes cotransfected with the human CD4 and chemokine receptors. Entry of the M-tropic (YU2) HIV-1 pseudotype was shown to be mediated by either CCR3 or CCR5, the T-tropic (HXB2) HIV-1 pseudotype by CXCR4, and the dual-tropic (89.6) HIV-1 or the SIVpbj1.9 pseudotype by CCR5, CCR3, or CXCR4. Our data indicate that the mechanisms for HIV-1 entry are both receptor-specific and cell type-specific, and that chemokine receptor expression on AM does not fully explain cell susceptibility to different virus isolates.